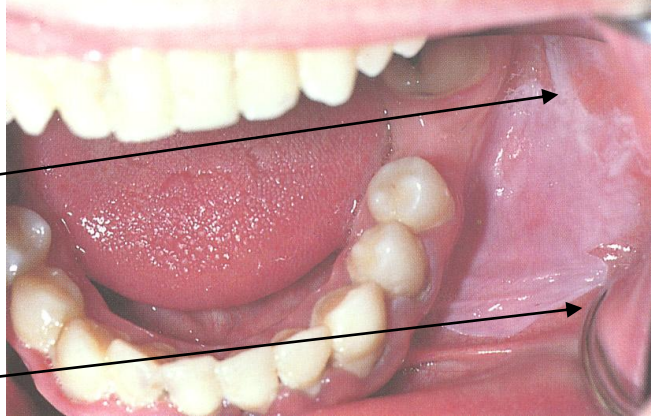


Oral ulceration and vesiculobullous diseases lab: chemical burns:

Aspirin burn:

- Homogenous lesion from the sulcus to the middle of the buccal mucosa.
- White lesion (plaque) caused by necrosis (completely burnt).
- It sloughs from the necrotic side leaving ulceration and may be not sloughed in the other side (oedema).



- It is due to usage of aspirin as a local obtundant for the relief of toothache.
- It is dose and time related lesion.
- It is not tobacco pouch keratosis as it should be rough surface with thicker areas not necrotic..

- Here pt placing other materials; it could be tobacco keratosis as it has rough surface and could be low concentrated aspirin with chronic usage.



Hydrogen peroxide burn:

- In periodontal clinic, gingival sulcus could be irritated with hydrogen peroxide
- They use it as disinfectant material- If it is concentrated --- burn the surrounding tissue --- whitish area with necrosis and sloughing.



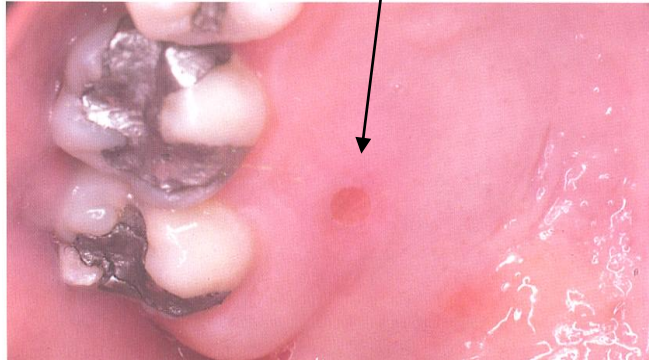
Formocresol burn:

- Formocresol is a material used in pulpotomy in children and sometimes in adult if there is severe pain in irreversible pulpitis.
- This usage is not ideal, not used in the health centre but used in private practice.
- They use it to relief pain but if they apply large quantity it will leak out to the gingiva producing necrosis of gingiva and bone necrosis also if it leaks through the apical foramen.
- It should not be placed in high concentration or for long durations



Anesthetic necrosis:

- Palatal injection or paraesthesia may induce focal area of necrosis due to:
 1. Increased fluid injected in the submucosa that compress blood supply and induce necrotic area.
 2. Adrenaline is in high concentration in the local anesthesia inducing vasoconstriction --- Reduced blood supply --- necrosis.
- Most likely the submucosa is very little and there is no area for anesthetic solution to spread.



Factitious ulcer:

- Areas of ulceration, thick whitish areas spreading all over the buccal mucosa not only at occlusal plane.
- It this whitish areas can't be removed by scrapping (even if some flakes can be removed),



starts chewing thickened epithelium



- it is habitual cheek biting.
- As it is low grade trauma over prolong period so the tissue will have time to induce keratosis and thickening of the epithelium --- pt so it will get these sloughs.

Note:

It is not likely to be pseudomembranous candidosis as there are areas of ulceration but in pseudomembranous candidosis the dr. induce the ulcer by scrapping it removing the whitish pseudomembrane then you will see the red erythematous areas, but here ulcers are already found on the buccal mucosa. If the lesion can't be scrapped --- don't think about pseudomembranous candidosis at all.

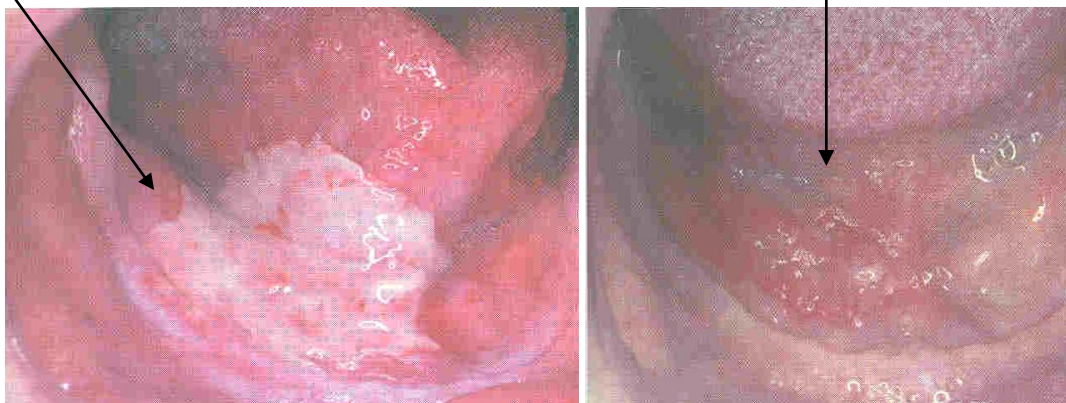
- Another example of self-induced ulcer:

Some pt use their finger nail (as a habit) to scratch the gingiva inducing ulcerations.



Radiation ulcer:

- Trauma to the tissue due to radiation.
- Radiation in area of SCC this is erythroplakia that turn to be SCC.
- After radiation in the same area whitish membrane, some redness, may be confused with recurrent tumor.



Recurrent aphthous stomatitis; RAS:

Minor type:

- Pt with orthodontic appliance which is a source of trauma and there is an ulcer with the typical appearance of aphthous ulcer which is having whitish pseudomembrane with red area at periphery.



- Traumatic ulcer usually red with sloughed surface caused by cheek biting but aphthous ulcer has whitish pseudomembrane with red margin.

- Common findings in these three minor ulcers:
 - 1- less than 10 mm in diameter
 - 2- they are 1-5 in number
 - 3- shallow ulcerations with whitish pseudomembrane, gray- yellow base with erythematous margin
 - 4- Affect non-keratinized mucosa.



Diagnosis:

RAS exacerbated by orthodontic appliance.

Heal without scarring within 10 days as it is minor



**Remember:

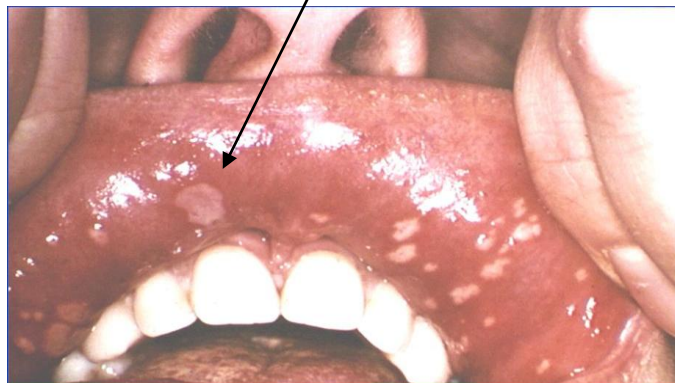
Trauma is one of the co-factor of aphthous but the aetiology is immunology or idiopathic.

Trauma may increase the recurrence or increase the number of ulcers or even modify the shape.

In the first picture there are rough irregular borders but may be from the orthodontic appliance but since there are other ulcers then it is for sure not traumatic.

Herpetiform aphthous:

- More than 10 ulcers on upper labial mucosa (multiple, small, pin head sized ulcers) occur on any part of the oral mucosa.
 - Heal within 2-3 weeks and recur in duration less than 1 month.
- A- With scar if small ulcers form a big white area of ulceration as they clustered together giving irregular outline.
- B- Without scar if they are pinpointed ulcers.
- Occur in elderly especially with nutritional or vitamin B12 deficiencies.



Major Aphthae:

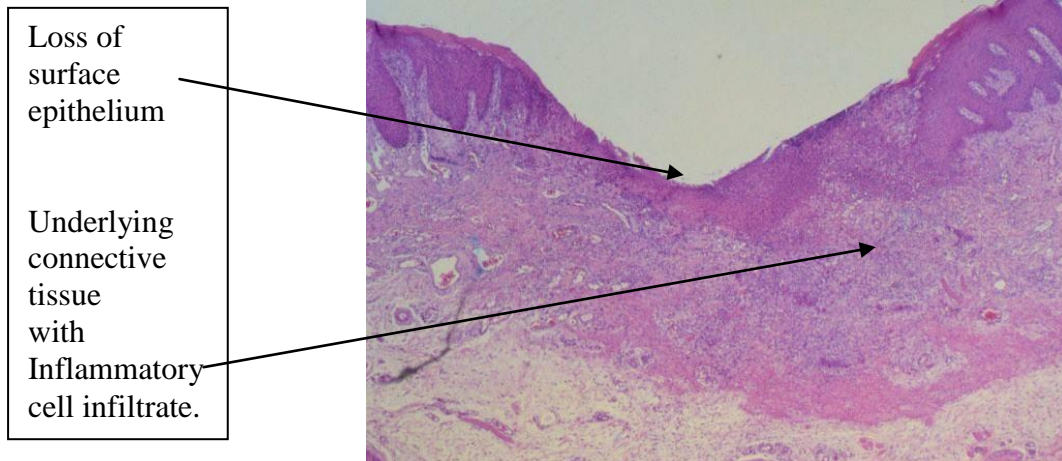
- More than 1 cm in diameter, 1-10 ulcers may be it is chronic as there is necrotic surface in the middle.

- It needs 4-6 weeks to heal with scarring.
- Signs of healing in the periphery and may be some fibrosis also on the periphery.
- Common sites: lips, soft palate, tonsillar areas and oropharynx.
- So chronic big ulcer on the soft palate, it could be:



- 1- Major aphthous ulcer
- 2- Deep fungal infection
- 3- SCC

- This slide describe the histo- pathological features of aphthous ulcer:



Inflammatory cell infiltrate:

- It is CD4+ (T-helper/inducer) in the beginning --- CD8+ (T-suppressor/cytotoxic) in the ulcerative phase --- CD4+ again in the healing phase.

Behçet's syndrome:

- Cutaneous pathergy test: redness and pus formation giving sterile pustule of the skin developed 1 day after injection with saline.
- Inflammation not this much but occur due to vasculitis associated with hyperactivity of polymorph neutrophils.



Figure 9-10 • Behçet's syndrome. Sterile pustule of the skin that developed 1 day after injection of saline. This reaction is termed cutaneous pathergy.

- There is genetic link with antigen HLA-B51 or caused by immuno-mediated mucosal damage.

- Big chronic ulcerative lesion on the tip of the tongue.
- It is exophytic, indurated looks attached to the underlying tissue.



Differential diagnosis:

Reactive trauma:

Reactive infection:

Neoplastic:

Idiopathic:

- *Traumatic ulcerative Granuloma
- *Factitial injury

Chronic viral infection: SCC

major aphthous ulcer

Herpes simplex
cytomegalovirus in
immuno- compromised pt.

Bacterial infection:

Oral tuberculosis but usually
it is on the dorsum of the tongue
not on the tip

Syphilis: chronic at areas of
entrance of the bacteria.

Fungal infection:

Deep fungal infection caused by
histoplasmosis, cryptococcosis, mucormycosis

Note:

Neoplastic could be connective tissue in origin when surface epithelium is ok or secondarily inflamed.

And it could be Epithelial in origin if there are epithelial changes.

Erythema multiformi:

- It has several clinical and microscopical forms.

- Target lesions on skin:
Central red bulla --- white edema --
- red bulla

So centric rings of varying erythema and edema, the centre may be intact or ruptured and crusted bulla.



- Intra- orally:
Irregular ulceration spreading all over the buccal mucosa with discomfort.
Biopsy from the lesion or peri-lesional area show sub or intra epithelial separation and epithelial necrosis.



Note:

If this peri- oral lesions are the 3rd or 4th recurrence it can't be primary herpes simplex infection as peri-oral vesicles, crusting and gingival ulcerations in primary HSV infection should occur for the first time to be called primary.



To be sure it is viral infection or hypersensitivity reaction to certain medications; take a blood sample searching for Ig against HSV.

Pemphigus vulgaris:

- Desquamative gingivitis which is ulcerative lesion on attached gingiva.
- It can't be ANUG as there is no grayish- green pseudomembrane on marginal gingiva and no loss of inter dental papilla which are the key features for the ANUG.



- And it is not AIDS related gingivitis as it is not linear erythema due to hypersensitivity reaction to Candida without pseudomembrane or necrosis like ANUG.



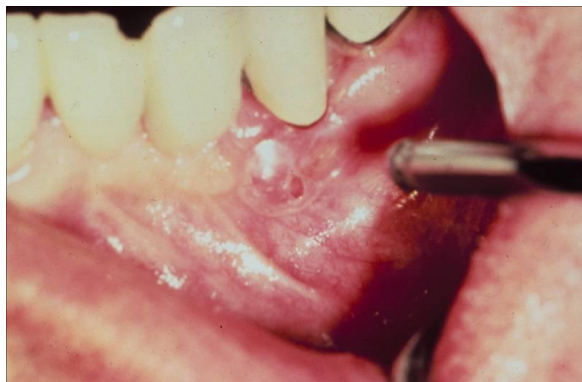
- Another picture for the same case.

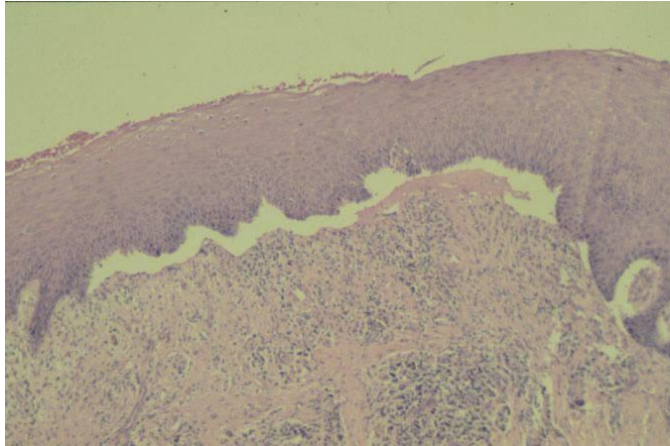
Differential diagnosis:

- 1- pemphigus vulgaris
- 2- pemphigoid
- 3- Erosive lichen planus.
- 4- Linear Ig A disease.
- 5- Allergy to medication, mouth wash, certain flavor or topical material.

Nickolsky sign:

It is gentle lateral rubbing or pressure on non- affected mucosa apparently normal --- within 2 min --
- pt still in the clinic --- bullous formation will be induced in pemphigus and pemphigoid.





Take a biopsy:
There is sub- epithelial separation.

Differential diagnosis:

- 1- Mucous membrane pemphigoid.
- 2- Linear Ig A disease.
- 3- Epidermolysis bullous acquisita.

Note:

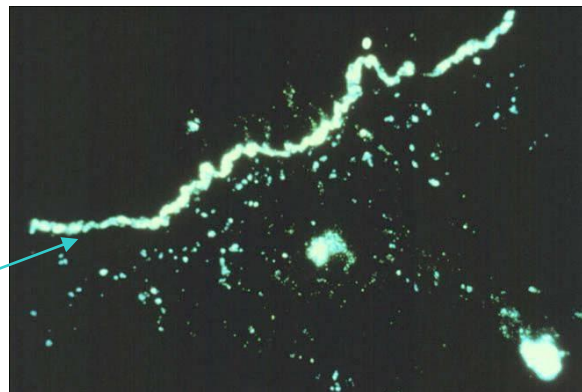
- It is clear separation so not erythema multiformi.
- The difference between mucous membrane pemphigoid and Epidermolysis bullous acquisita is that the target in the first is the hemi-desmosomes of the basement membrane but in the second one is the anchoring fibrils below hemi- desmosomes which interacts with the underlying collagen fibers in the lamina propria.

DropBooks

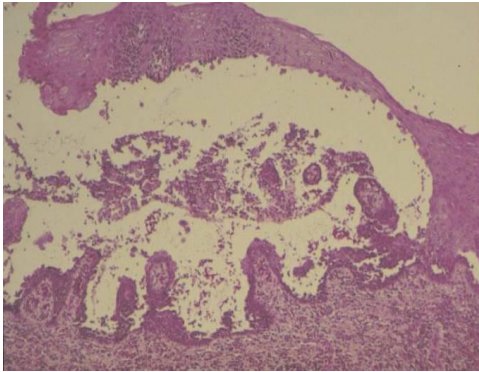
Mucous membrane pemphigoid:

- Here we have Ig G deposition as linear band at the basement membrane --- Mucous membrane pemphigoid.

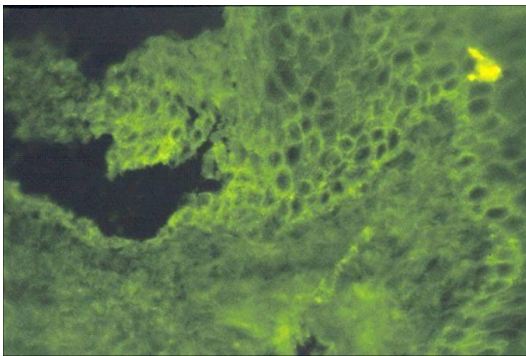
Ig G deposition and C3



Pemphigus vulgaris:



- Zank cell which looks small and rounded in the pemphigoid.
- Connective tissue and intra-epithelial separation.



- In direct immuno-fluorescent: Loss of desmosomes giving fish net appearance circling the boundaries of the cells only.

Dermatitis herpetiformis:

- A pt having dermatitis and may be intra-oral ulcerations and may be not but mainly dermatitis.



- In immuno-fluorescent: This is the surface epithelium and down there is connective tissue --- discontinuous or granular deposition of Ig A.

(fluorescence -
no fluorescence)

Linear Ig A disease:

- Eruption on the skin, dermatitis, and intraoral ulcerations.



- Direct immuno-fluorescent shows continuous homogenous band of Ig A.

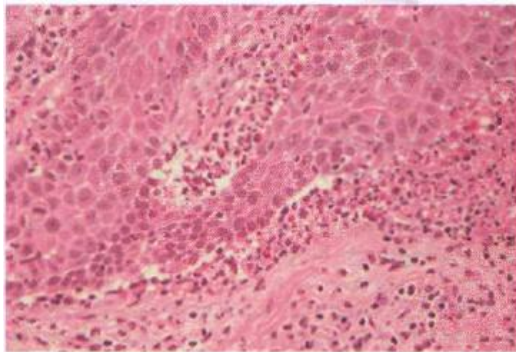
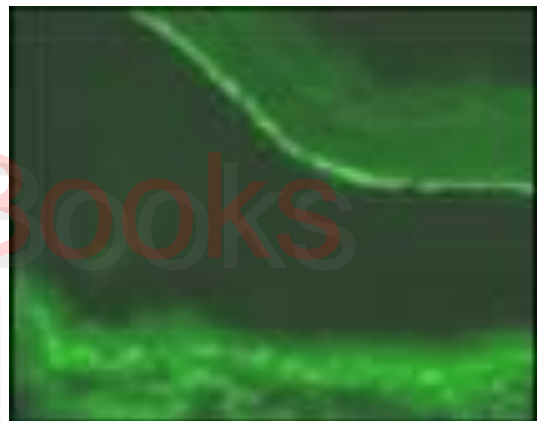


Figure 1-30 Linear IgA disease showing subepithelial separation with neutrophils and eosinophils.

- sub-epithelial separation with a lot of neutrophils and some eosinophils.

Epidermolysis Bullosa and Epidermolysis Bullosa Acquisita:

- Epidermolysis Bullosa is a condition starts since birth, with genetic analysis mutated genes could be found.
- In the areas of trauma, there is always fibrosis and limited movement.



- This pt is from the simplest type which is not dystrophic or recessive but compatible with life. The rest of her sisters and brother (13 children), all of them died as their cases were not compatible with life.



- She had fused fingers due to continuous trauma and there are scars all over her hands.

- She had limited mouth opening with pale whitish scars with ulcers on the palate and whitish areas. She complained from severe pain.
- The tongue is like a mass of scar could be due to biting; it does not even look muscular.
- There is bleeding, ulcerations.



In the clinic endodontic specialist said no way to do RCT with this limited mouth opening. Some suggested extracting the most painful teeth. So the end result will be complete denture.

The Acquisita type:

- Same clinically but it starts to appear in the twenties or thirties in the adulthood as the body starts making antibodies against collagen 7 or anchoring fibrils.



- So with continuous trauma --- scars --- fusion
Abnormal epithelium --- easy sloughing --- abnormal healing.

Angina Bullosa Haemorrhagica (oral blood blister):

- It is spontaneous blood filled Bullae (blisters) on any part of the oral mucosa, mostly on the soft palate.
- Solitary, in middle aged or elderly pts.
- 2-3 cm in diameter.
- Early perforation leaving ulcer heals uneventfully.
- Sub-epithelial Bulla with separation within the basement membrane zone.
- No Igs --- No Abnormalities in the blood coagulation or in the tissue.
- Could be due to trauma or steroids inhalers.

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